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Transparency declarations
None to declare.

Supplementary data
Table S1 is available as Supplementary data at JAC Online (http://jac.oxfordjournals.org/).

References

Sir,
Further to the case described by Pinto and Merlino in their recent correspondence, we report our experience of an outbreak of CO₂-dependent methicillin-resistant *Staphylococcus aureus* (MRSA) on a UK regional liver transplant unit (RLU). Six patients and one staff member were infected or colonized with a strain of MRSA, which would not have been reliably detected by routine screening methods. The laboratory characteristics of this strain, infection control strategies and the implications for screening are discussed.

In early 2008, a liver transplant recipient was screened for MRSA on admission to the critical care unit from the RLU. Small poorly growing green colonies were isolated from a nasal swab on a chromogenic MRSA identification medium (chromID Small). The colonies were slide-coagulase positive (SlideStaph-Plus reagent; bioMérieux). The strain repeatedly failed to grow after 24 and 48 h of aerobic incubation for 24 h. The colonies were slide-coagulase positive (SlideStaph-Plus reagent; bioMérieux). When subcultured onto BA and incubated overnight at 37°C in air plus 5% CO₂, however, a heavy growth of an isolate with typical *S. aureus* colony morphology was observed. Susceptibility testing performed on IST according to BSAC guidelines, but in CO₂-enriched atmospheric conditions (5% CO₂), confirmed resistance to cefoxitin, erythromycin, clindamycin, moxifloxacin and trimethoprim.

A possible outbreak was suspected as the microbiologist and infection prevention and control team were aware of another liver transplant recipient on the RLU who was found, 3 weeks earlier, to be colonized with a strain of MRSA that grew much better in 5% CO₂ than aerobically. This index case was a male patient in his 60s, 5 months into an admission for decompensated alcohol-related liver disease. MRSA was not detected from his routine admission screening swabs. Following an orthotopic liver transplant his post-operative course was complicated by acute rejection, hospital-acquired pneumonia and *Clostridium difficile*-associated diarrhoea. The following month he suffered an acute clinical deterioration and underwent an exploratory laparotomy. MRSA, as described above, was isolated from enrichment culture of intra-abdominal tissue. Repeat superficial swabs from this patient were incubated in CO₂-enriched atmospheric conditions, demonstrating carriage of the organism at multiple clinical sites. Despite topical decolonization attempts, throat and perineum swabs remained positive at discharge and also at follow-up 4 weeks later.

Following the identification of the second case, all patients and staff on the RLU were screened for carriage of CO₂-dependent MRSA using chromID MRSA medium in 5% CO₂. A deep clean of the ward was carried out, and infection prevention and control practices were reinforced. Environmental screening swabs prior to the deep clean and post-decontamination were negative. Four further cases (three patients and one staff member—a student nurse) were found to be colonized at one or more sites by a CO₂-dependent strain of MRSA. Ongoing targeted screening revealed a seventh case 5 weeks after the initial outbreak. This patient was admitted to the RLU during March 2008, but had been discharged 2 days prior to the recognition of the outbreak and screening swabs were found to be positive when he was readmitted to the RLU in May 2008.

Table 1 summarizes the admission date and location of hospitalization for each case, along with the site of first isolation of MRSA and the length of time on the unit when MRSA was first isolated.

Molecular testing was performed locally at the Institute of Cell and Molecular Biosciences, University of Newcastle upon Tyne. PFGE showed that strains generated identical profiles to that of EMRSA-15, except for one strain in which a single band was reduced in size. Diagnostic PCR was used to detect the presence of the *mecA* and *Panton–Valentine leucocidin* (PVL) genes. All strains were *mecA* and *mecA* positive, but PVL negative. Multilocus sequence typing and staphylococcal cassette chromosome mec (SCCmec) typing indicated that the strains were MRSA ST22 with SCCmec type IV, confirming that they were variants of EMRSA-15.

Following prompt recognition, this outbreak was successfully brought under control by reinforcement of good infection control practices. Despite its unusual growth characteristics, the strain was identified as a clone of EMRSA-15, the predominant UK epidemic strain. Transmission of CO₂-dependent *S. aureus* has been previously described in the literature as early as 1955. Gómez-González et al. recently observed nosocomial transmission of a strain of *S. aureus* that was auxotrophic for CO₂, and described the clinical and molecular characteristics of such small-colony variants of *S. aureus* in their institution. To our knowledge, ours is the first reported outbreak of a CO₂-dependent MRSA. Similar outbreaks may be missed if screening

<table>
<thead>
<tr>
<th>Case</th>
<th>Admission date</th>
<th>Length of time on unit (days)</th>
<th>Length of time on the unit when MRSA first isolated (days)</th>
<th>Site of colonization or infection first detected</th>
<th>Location on RLU</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>Sep 2007</td>
<td>217</td>
<td>168</td>
<td>intra-abdominal tissue</td>
<td>cubicle</td>
</tr>
<tr>
<td>2</td>
<td>Feb 2008</td>
<td>15</td>
<td>15</td>
<td>nasal swab</td>
<td>cubicle</td>
</tr>
<tr>
<td>3</td>
<td>Feb 2008</td>
<td>28</td>
<td>28</td>
<td>abdominal drain site</td>
<td>bay A</td>
</tr>
<tr>
<td>4</td>
<td>Mar 2008</td>
<td>33</td>
<td>17</td>
<td>abdominal drain site</td>
<td>bay A</td>
</tr>
<tr>
<td>5</td>
<td>NA</td>
<td>NA</td>
<td>NA</td>
<td>nasal swab</td>
<td>staff</td>
</tr>
<tr>
<td>6</td>
<td>Mar 2008</td>
<td>13</td>
<td>13</td>
<td>nasal/perineum swab</td>
<td>cubicle</td>
</tr>
<tr>
<td>7</td>
<td>Mar 2008</td>
<td>2</td>
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<td>NA</td>
<td>bay A</td>
</tr>
<tr>
<td>8</td>
<td>May 2008</td>
<td>2</td>
<td>0</td>
<td>nasal swab</td>
<td>NA</td>
</tr>
</tbody>
</table>

NA, not applicable.
swabs are processed by conventional methods. Establishing the local prevalence of CO₂-dependent MRSA is necessary to determine whether targeted screening is required.

Acknowledgements
The main findings were presented in abstract form in a poster (P855) at the European Congress of Clinical Microbiology and Infectious Diseases, Helsinki, 2009.

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References

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Cross-border transmission of OXA-48-producing Enterobacter cloacae from Morocco to France

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Sir,
The blaOXA-48 gene is plasmid-borne and encodes a carbapenem-hydrolysing class D β-lactamase that was first identified in Klebsiella pneumoniae in Turkey in 2003.¹ This enzyme confers resistance to penicillins and reduced susceptibility to carbapenems, but spares expanded-spectrum cephalosporins.¹ It has been identified in multidrug-resistant isolates, which often accumulate multiple resistance mechanisms, including production of extended-spectrum β-lactamases (ESBLs).² The high prevalence of OXA-48 producers in Turkey has been demonstrated, but there are additionally scattered reports of OXA-48-producing K. pneumoniae in several countries, such as Belgium, France and Lebanon.³ Two recent reports, one of OXA-48-producing Escherichia coli and K. pneumoniae isolates in Tunisia⁴ and the other of OXA-48-producing K. pneumoniae from Morocco,⁵ suggested the widespread nature of OXA-48 in North Africa. It is noteworthy that many OXA-48 producers co-express ESBLs, but several OXA-48-producing isolates that do not carry ESBL genes may remain susceptible to broad-spectrum cephalosporins.³

We report now the identification of blaOXA-48-positive Enterobacter cloacae isolates recovered at two hospitals in two different cities in France from patients who had been transferred from Morocco, where they had been hospitalized.

E. cloacae isolate 501 was recovered in August 2010 from the urine and rectal swabs obtained from a female patient on her admission to the University Hospital of Saint-Etienne, south-east France. She had been transferred from the intensive care unit (ICU) of the Hospital of Fez, Morocco, where she had been admitted after a traffic accident. She was not considered as infected and thus was not treated with any antibiotic. Isolate 501 was resistant to penicillins and cephalosporins, and had decreased susceptibility to carbapenems, with MICs of imipenem, ertapenem and meropenem of 1, 16 and 1.5 mg/L, respectively. It was therefore in the non-susceptibility range according to the updated CLSI guidelines (June 2010)⁶ for ertapenem and meropenem, but not for imipenem. According to the EUCAST breakpoints (www.eucast.org/) it was only resistant to ertapenem. Isolate 501 was also resistant to tigecycline, gentamicin, netilmicin, sulfonamides, tetracycline, tigecycline, chloramphenicol, rifampicin, trimethoprim, nitrofurantoin, fosfomycin and fluoroquinolones, and remained susceptible only to amikacin and colistin.

In August 2010, another patient was admitted to the Bicêtre Hospital, in a suburb of Paris, France, having been transferred from the Hospital of Agadir, Morocco, where he had been hospitalized after a stroke. This patient was admitted to the ICU at the Bicêtre Hospital, and he had not received any previous antibiotic treatment. Rectal screening on his admission resulted in the growth of a multidrug-resistant E. cloacae. Isolate BOU was resistant to penicillins and cephalosporins and had decreased susceptibility to both imipenem and meropenem, with MICs of 1.5 mg/L, being therefore in the non-susceptibility range...